Armed Forces College of Medicine

AFCM

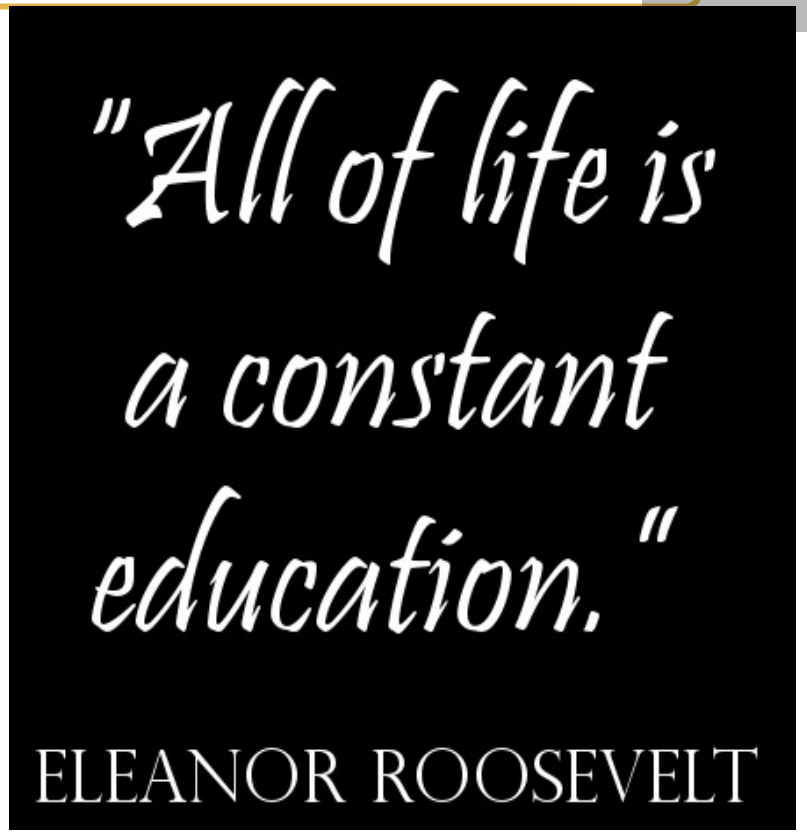
Cardiopulmonary module

Clinical Integrated Cases

Session 2

Basic Science Principles for Clinical Reasoning

1- Critical thinking
2- Introduction to common forms of disease
3- Apply basic knowledge in a realistic clinical scenario.
4- Interpersonal skills-(share and apply your knowledge)… communication…patient safety
5- Life long self-directed learning



Case co-ordinators

Prof. Drs. Manal, Tamer, Bassant, Lamiaa

Sharing departments

- Cardiology department
- Histology department
- Microbiology department
- Pathology department
- Physiology department

Case: Dyspnea with fever

Dyspnea with fever

Soad is a 52-year-old woman. She is a housewife living in elmarg. She is married and has one child. She is known to have dyspnea on moderate exertion and was diagnosed as have rheumatic heart disease with mitral stenosis.

She was having dyspnea and lower limb edema on and off , which were relieved by medications.

Dyspnea with fever

In the last 5 days she started to complain of fever and dyspnea on mild exertion. She had loss of appetite, malaise, muscle pain and arthralgia
Her husband noticed that she gets **dyspneic after minimal efforts**

The doctor asked about recent infections and Soad said that she **removed a tooth 2** weeks before the start of the symptoms

Past History

Soad has a history of anemia attributed to menorrhagia

Dyspnea with fever and chills

Physical examination revealed:

Pulse: 120beats/min, regular and equal on both sides

Blood pressure: 120/80mmHg

Respiratory rate: 24cycle/min

Temperature: 39.2ºC

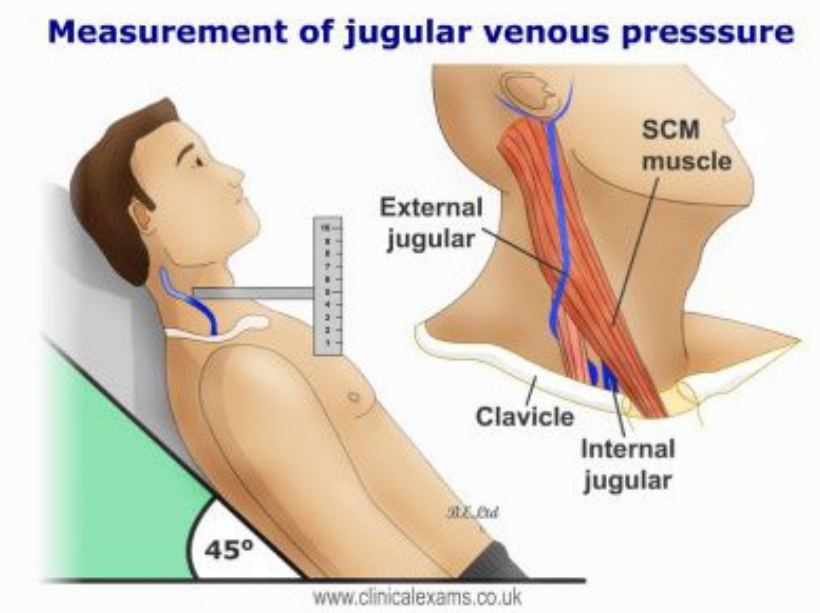
Pale skin and mucous membranes.

Conjunctival hemorrhage

No cyanosis, no jaundice,

no stigmata of hyperlipidemia

Neck veins: were **congested and pulsating**



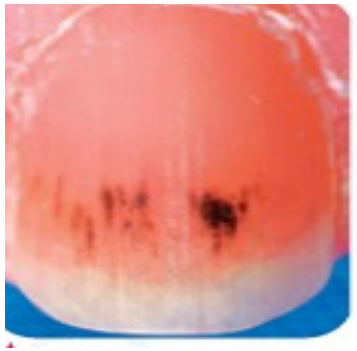
http://www.clinicalexams.co.uk/elevated-jugular-venous-pressure-jvp/

Dyspnea with fever and chills

<u>Physical examination revealed</u>:

Mouth examination

Dental caries and poor oral hygiene



Nails: splinter hemorrhages

Skin:
Peticheal hemorrhages
Tender subcutaneous nodules

Abdominal examination:
Splenomegaly

1- What is your provisional diagnosis?

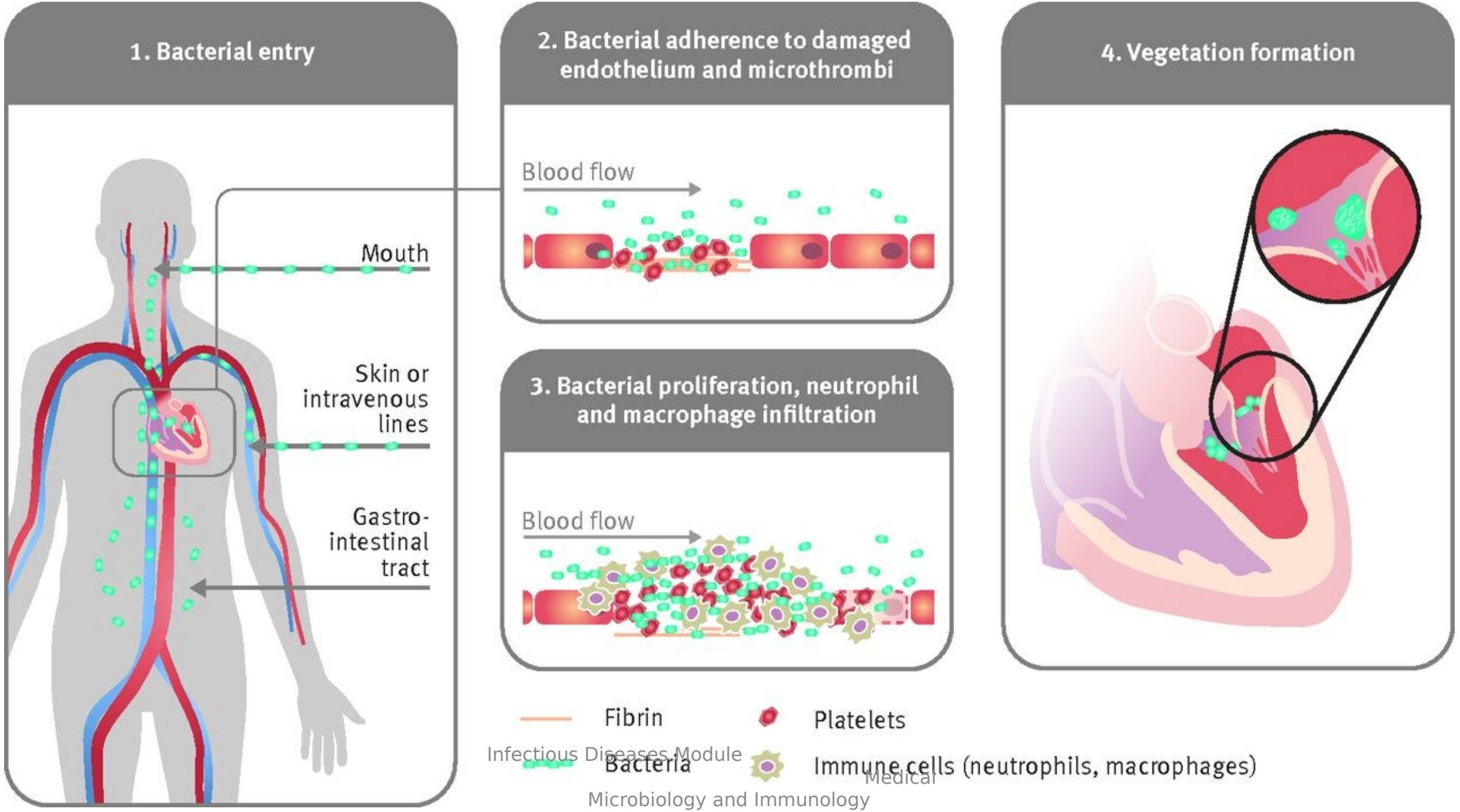
Justify your answer

- A known case of **rheumatic heart disease** who has history of **tooth extraction** one week before presentation, she presented with **fever** and **worsening dyspnea.**

- fever in a cardiac patient should alert us about either infective endocarditis or rheumatic activity or any other infection.

- Given the history of tooth extraction (**source of bacteria in the blood**) so we should keep in mind infective endocarditis.

2- What is the significance of the history of rheumatic heart disease and tooth extraction in Soad’s condition?


1. Bacterial entry
Mouth
Skin or intravenous lines
Gastro-intestinal tract
2. Bacterial adherence to damaged endothelium and microthrombi
Blood flow
3. Bacterial proliferation, neutrophil and macrophage infiltration
Blood flow
4. Vegetation formation
Fibrin
Platelets
Bacteria
Immune cells (neutrophils, macrophages)

- Hematogenous spread of flora following dental surgery ..etc.

- Circulating bacteria colonize **damaged** valvular endothelium.

- Adhesion of bacteria to the damaged endothelium is enhanced by their ability to produce a **glycocalyx.**

- Once the infection has begun, a combination of

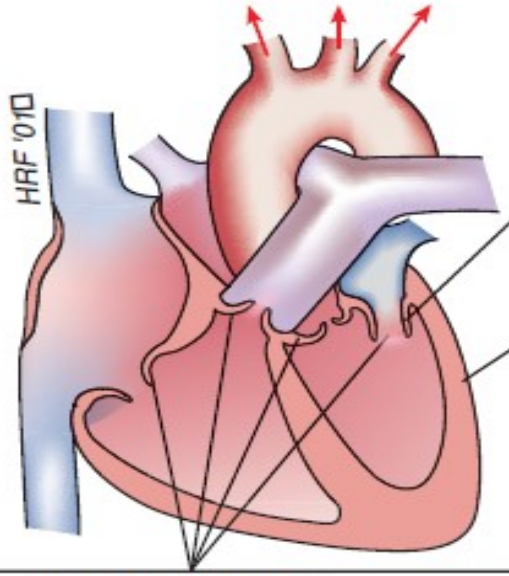
What are the common organisms that cause this condition?

Cardiovascular System Infections

K. pneumoniae (38)
Enterobacter (38)

P. aeruginosa (45)
B. fragilis (50)

S. pyogenes (17)
S. aga



1- Acute endocarditis caused by highly virulent organism as *Staphylococcus aureus* .

2-*S*ubacute endocarditis caused by low virulent organism as *strept. viridans*.

B. burgdorferi (62)

ENDOCARDITIS

Native Valve	IV Drug Users (often tricuspid)		Prosthetic Valve	
Viridans streptococci (23)	*S. aureus* (13)	Bacterial	*S. epidermidis* (14)	Acute
S. bovis (21)	Streptococci		*S. aureus* (13)	
S. pyogenes (18)	*E. faecalis* (20)		Gram– bacilli	
E. faecalis (20)	*P. aeruginosa* (45)		*C. albicans* (119)	
S. aureus (13)	*C. albicans* (119)	Fungal	Streptococci	Subacute
S. epidermidis (14)				

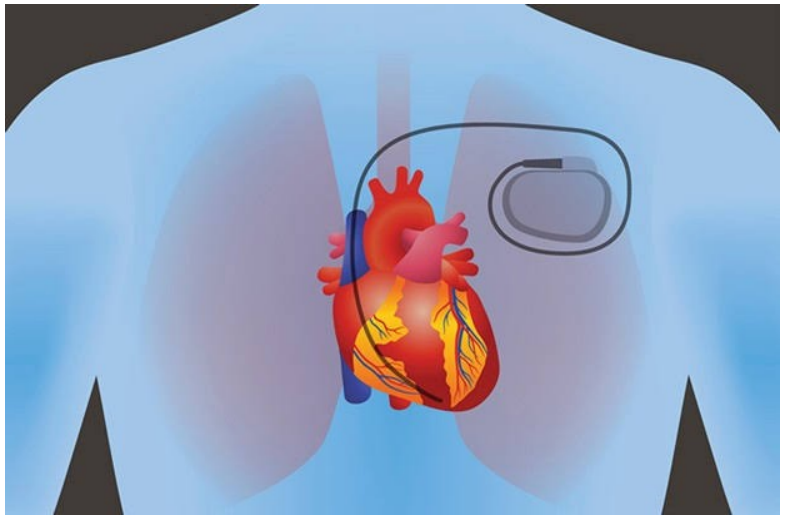
TABLE 71–1 Etiology of Endocarditis by Category

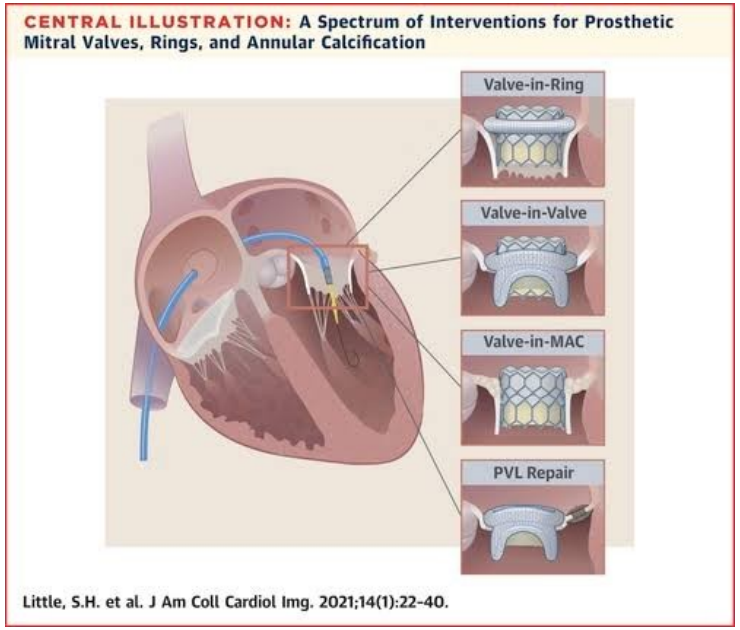
Category	Pathogen
Native valve	
Community onset	Viridans group streptococci, *Staphylococcus aureus, Streptococcus bovis, Enterococcus* species
Health care associated	*S. aureus, Enterococcus* species, *Staphylococcus epidermidis*
Intravenous drug user	*S. aureus*, gram-negative rods such as *Pseudomonas, Candida* species
Prosthetic valve	
Early	*S. epidermidis, S. aureus*
Late	*S. aureus*, viridans group streptococci, *Enterococcus* species, *S. epidermidis*
Pacemaker or defibrillator	*S. epidermidis, S. aureus*
Culture negative	Prior antibiotics, *Bartonella* species, *Coxiella burnetii, Brucella* species,

3- What are the risk factors of subacute infective endocarditis?

1. Congenital heart disease
2. Rheumatic valvulitis
3. Artificial valves
4. Percutaneous valve intervention
5. Pace maker wires

Devices




CENTRAL ILLUSTRATION: A Spectrum of Interventions for Prosthetic Mitral Valves, Rings, and Annular Calcification
Valve-in-Ring
Valve-in-Valve
Valve-in-MAC
PVL Repair
Little, S.H. et al. J Am Coll Cardiol Img. 2021;14(1):22-40.

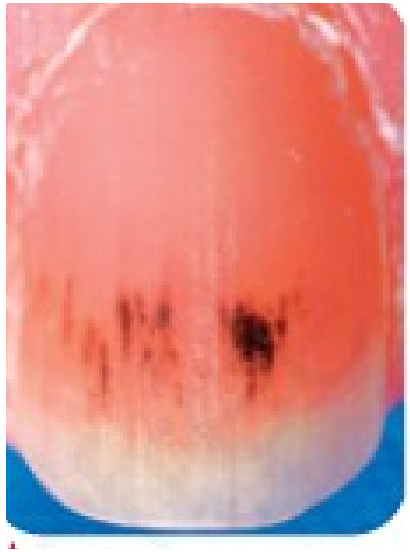
ck veins of Soad were congested and pulsating

Identify the vein the doctor is examining in Soad?

y do we examine neck veins <u>on the right side</u> ?

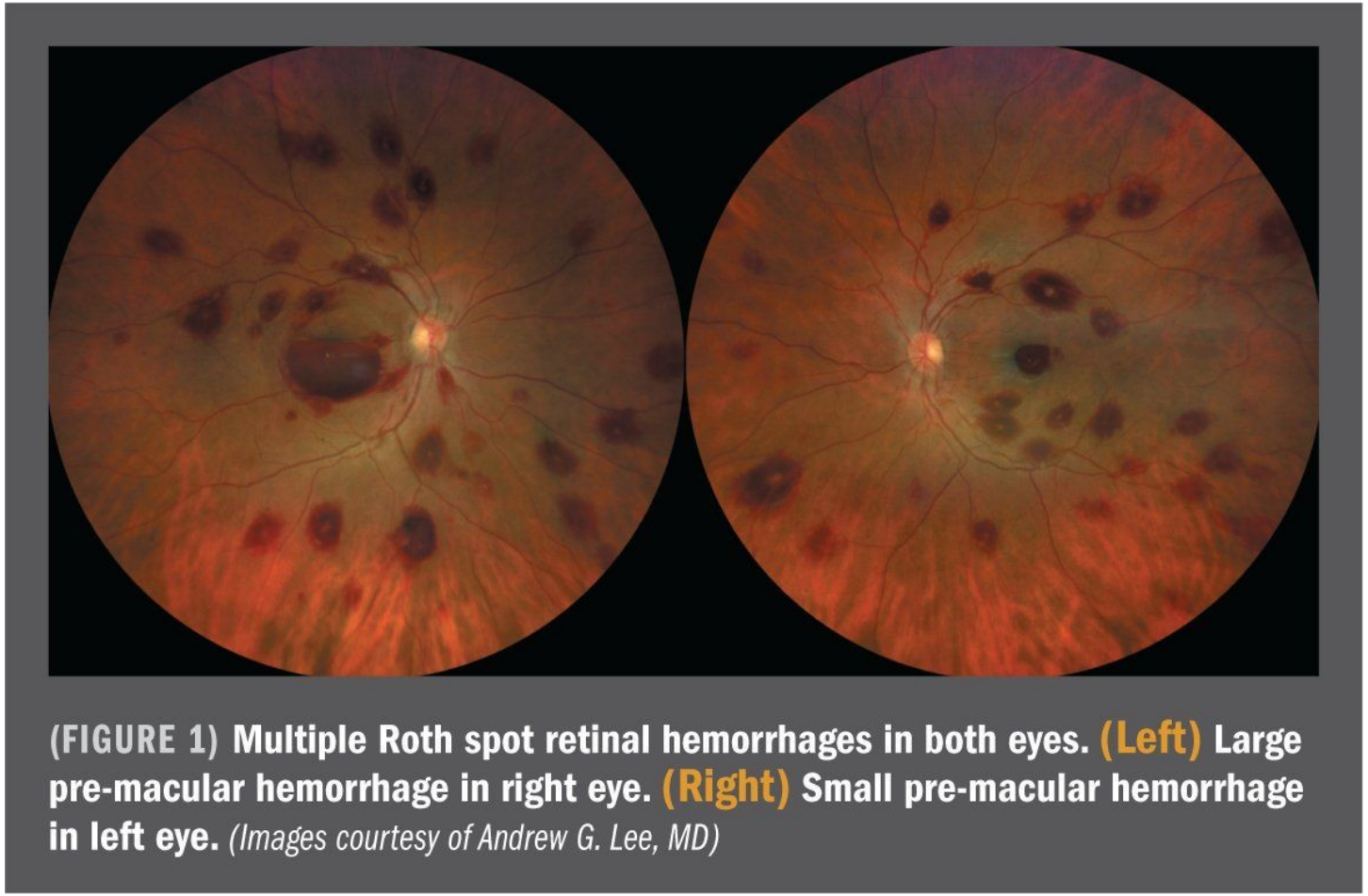
4- Explain skin and nail manifestations seen in Soad

- Splinter hemorrhages : dark red lines under the nail due to **septic emboli** leading to rupture of the tiny capillaries.
- Oslers nodes are tender small nodules affecting distal ends of fingers and toes due to either **immune complex deposition** in the distal small blood vessels or **septic emboli**.
- Janeway macules in the palms and soles mainly , due to micro emboli and thrombosis of the small blood vessels.



What can be found in fundus examination in this case?

Roth spots which are pale centered hemorrhagic spots in fundus due to micro emboli.



(FIGURE 1) **Multiple Roth spot retinal hemorrhages in both eyes. (Left) Large pre-macular hemorrhage in right eye. (Right) Small pre-macular hemorrhage in left eye.** *(Images courtesy of Andrew G. Lee, MD)*

5- What investigations are needed to confirm your diagnosis?

- **Blood cultures**
- **CBC with differential count**
- **CRP**
- **ESR**
- **Urine analysis, kidney function test**
- **Echocardiography**

Welcome Back

Dyspnea with fever and chills

The patient was hospitalized **The doctor asked for the following investigations:**

- **Blood cultures**
- **CBC with differential count**
- **CRP**
- **ESR**
- **Urine analysis, kidney function test**
- **Echocardiography**

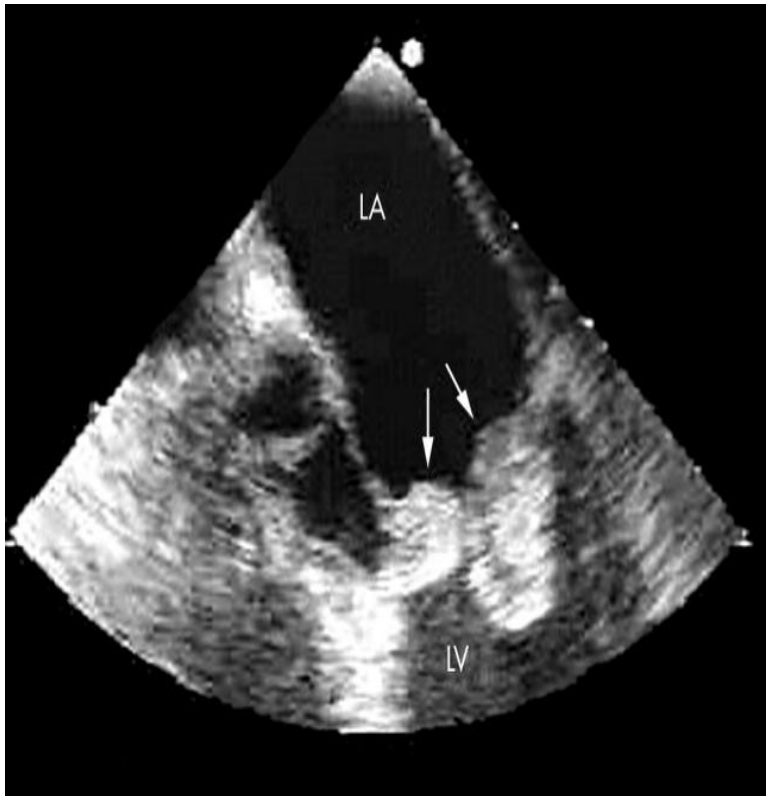
Dyspnea with fever and chills

Results showed:

- CBC: leucocytosis
- Blood culture showed streptococcus viridans in 2 cultures

- CRP: elevated
- ESR: elevated
- Urine analysis: normal
- Kidney function: normal

- Echocardiography showed vegetations on the mitral valve and newly diagnosed additional mitral regurge



Dyspnea with fever and chills

The doctor ordered:

IV Antibiotics first emperical till culture results available then change according to culture result

- **ESR: elevated**

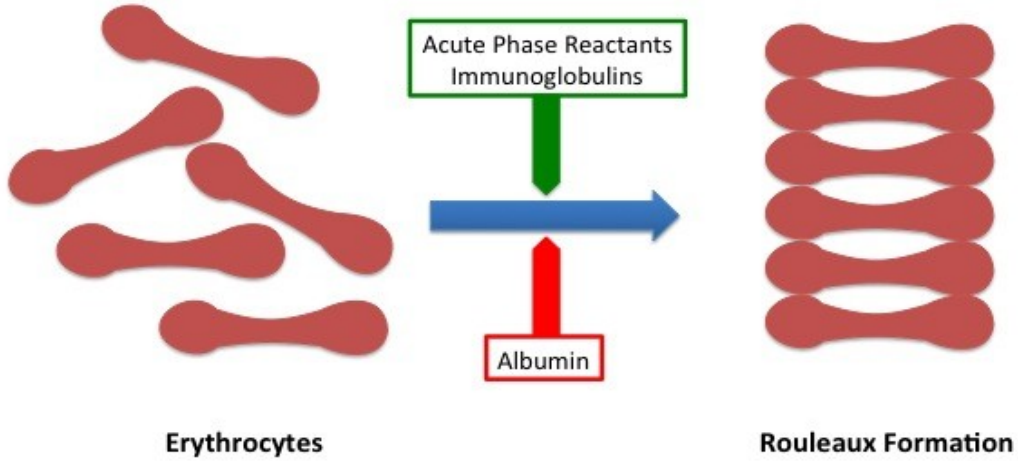
6- Explain the elevated ESR

Soad has Increased Immunoglobulins due to inflammation which decrease the negative charge of erythrocyte, so promotes formation of rouleaux and Rouleaux formation accelerates or increases ESR

ESR is not Diagnostic

If ESR above 100 mm

Chronic inflammation (eg.:TB)
Malignancy.
Collagen diseases



Rouleaux are stacks of erythrocytes. Their formation is formation is promoted by acute phase reactants and immunoglobulin and hampered by albumin. Rouleaux sediment faster than erythrocytes. The degree of rouleaux formation is one of the major determinants of ESR.

ESR:

Erythrocyte/plasma ratio
Level of fibrinogen & immunoglobulin

Factors that Increase ESR

- **Decreased Erythrocyte/plasma ratio:**
  e.g. Anemia. This change in erythrocyte-plasma ratio favors **rouleaux** formation which accelerate ESR.
- **Increase level of fibrinogen**
- **Increased Immunoglobulin**
  Both decrease the negative charge of erythrocyte, so promotes formation of rouleaux and increase ESR

Factors that decrease ESR

- **Increased Erythrocyte/plasma ratio**
  e.g. Polycythemia
- **Defibrinigenation:**
  Decreased fibrinogen decreases ESR

NB.: abnormal or irregular shape of RBC (Spherocytosis, Sickle cell) lower ESR

t is the microscopic structure of the endocardiu

The structure of the wall of the heart

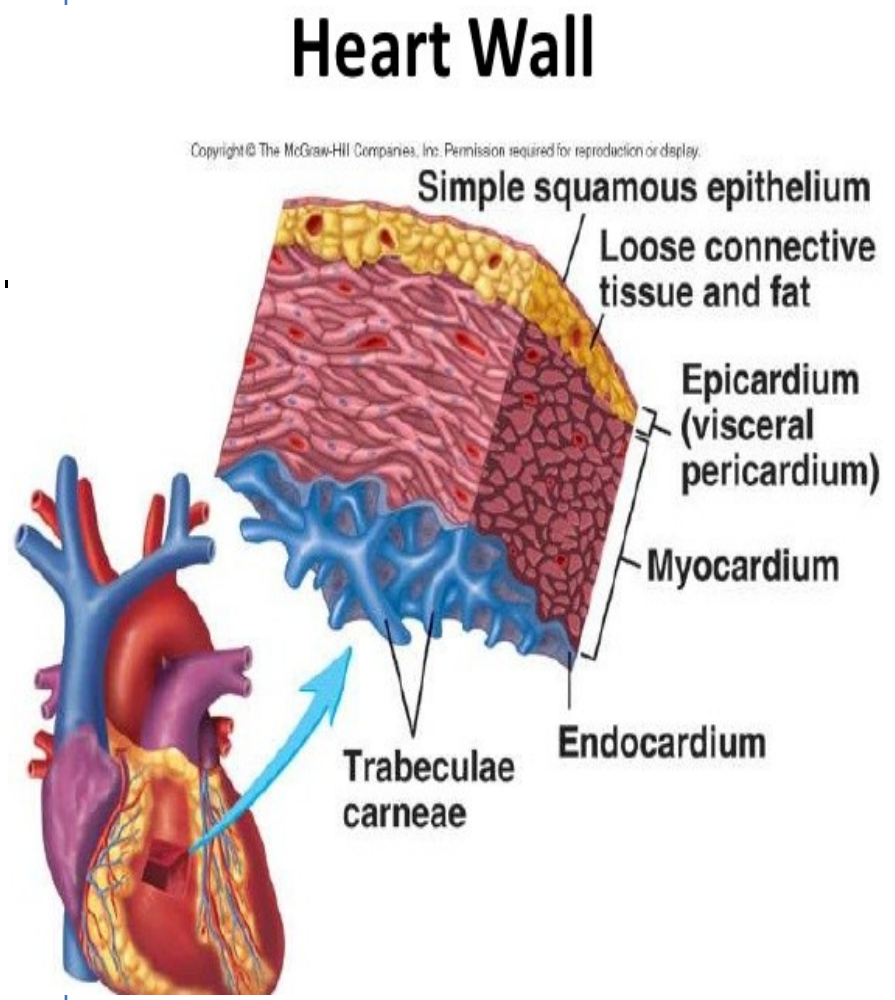
1. Epicardium: (visceral layer of pericardium):

Simple squamous ep. + C.T. contain fat & **branches of coronary arteries.**

2. Myocardium: cardiac m. run in different direction.

3. Endocardium: It lines the heart.

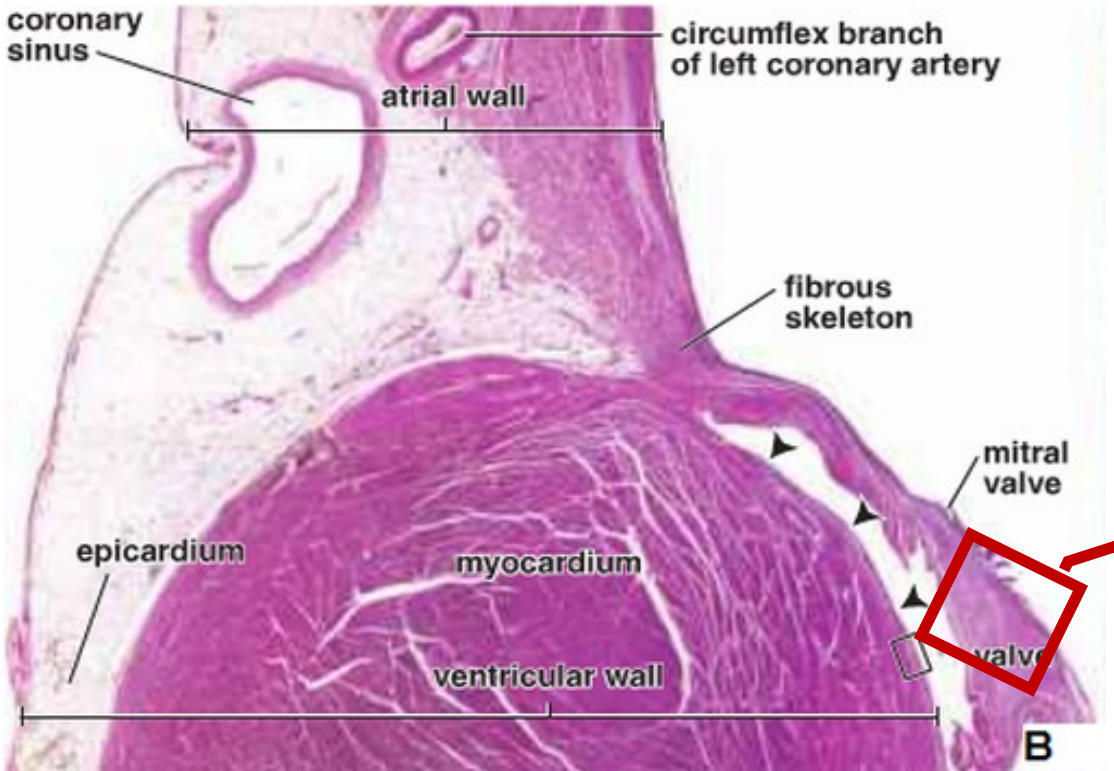
- Simple sq. endothelium & subendoth. CT
- Layer of dense C.T. contains elastic f.
- Layer of loose C.T. contains bl. V.& **Purkinje fibers.**



Valves of the heart

Valves are avascular

They are extension fold from endocardium ie. covered by endothelial cells on both sides,



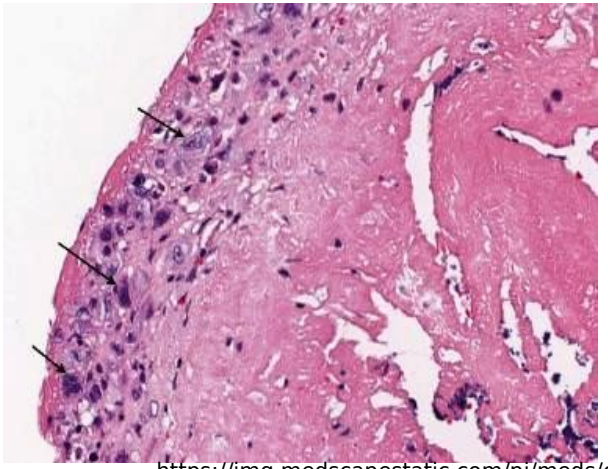
7- Describe the expected morphological changes of Soad’s mitral valve?

Subacutecute Infective Endocarditis

Pathological features:

1- Valve lesions:

- **Picture of the original disease**
- **Vegetations:**



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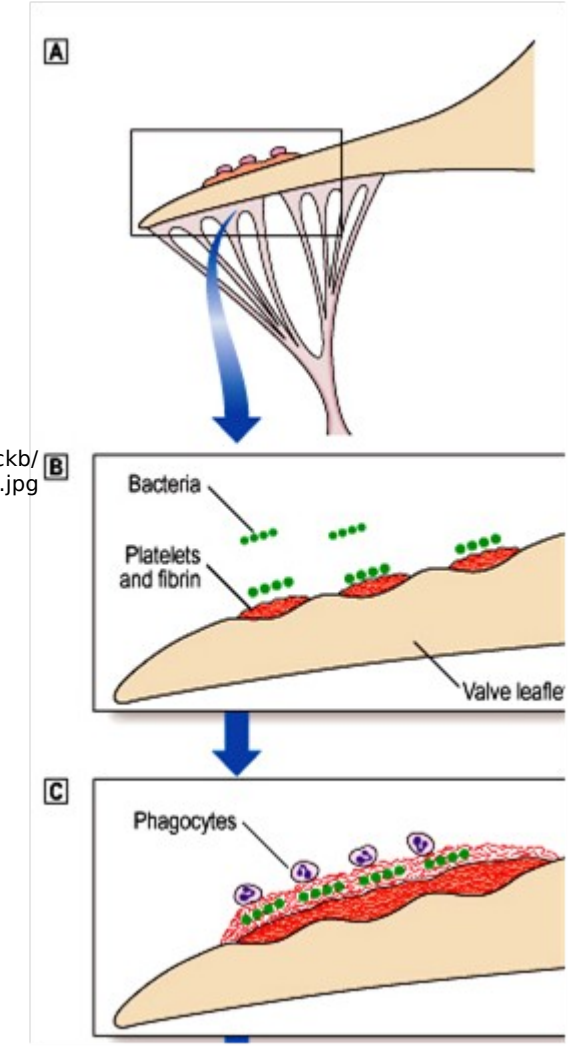


Grossly: Multiple, large, gray, friable, detachable, developing anywhere on the cusps & MacCallum's patch.

Microscopically: Vegetations consist of platelets + fibrin + bacteria + inflammatory cells (histiocytes)

8- The doctor asked Soad to make urine analysis and kidney function tests

What is the change that may occur in the following organs? (explain its pathogenesis)

- **The kidney**
- **The mesenteric artery**

Subacutecute Infective Endocarditis

Pathological features:

Embolic lesions:

- **Mycotic aneurysm: most common in cerebral & mesentric arteries → impacted embolus → mild inflamm → vessel wall fibrosis (weakening → dilatation aneurysm**

https://bilgiportali.info/wp-content/uploads/2019/01/anevrizma-nedir.jpg

Subacutecute Infective Endocarditis

Pathological features:

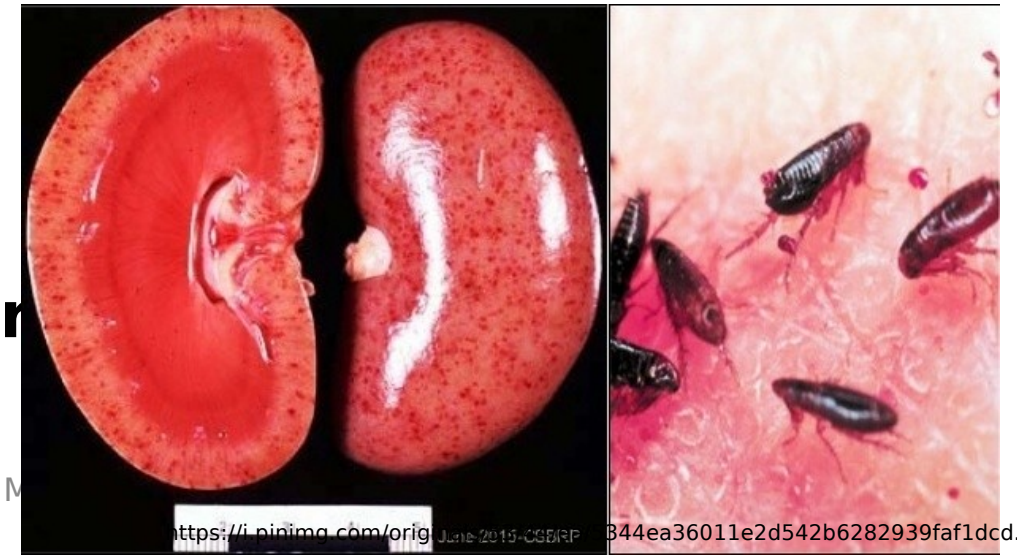
Toxic lesions: **Moderate toxemia:**

- **Focal glomerulonephritis: due to antibodies to bacterial antigens → Immuune complexes deposition → toxic capillaritis → focal glomerular necrosis & inflammation**

petechial hemorrhages

(flea bitten kidney)

Clinically: Hematuria & renal failur



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9- What are the complications?

- **Valve destruction** (incompetence, heart failure)
- **Septic embolization** for example to brain causing CVA
- Immune complex formation leading to **glomerulonephritis**

How will you manage Soad?

- **IV Antibiotics first empirical till culture results available then change according to culture result**

- **Treatment of heart failure.**
- **Surgical treatment sometimes is needed .**

Besides the treatment of acute condition , patient should be advised for antibiotic prophylaxis before surgical procedures.

Thank
you